CLINICAL PROCEEDINGS

of the

CHILDREN'S HOSPITAL

WASHINGTON, D. C.

April 1951

VOLUME VII NUMBER 5

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CLINICAL PROCEEDINGS

OF THE CHILDRENS HOSPITAL

13th and W Streets, Washington 9, D. C.

Vol. VII *April 1951* No. 5

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Published monthly by the Staff. Cases are selected from the weekly conferences held each Sunday morning at 11:00 A.M., from the Clinico-pathological conferences held every other Tuesday afternoon at 1:00 P.M., and from the monthly Staff meetings.

This bulletin is printed for the benefit of the present and former members of the Attending and Resident Staffs, and the clinical clerks of Georgetown and George Washington Universities.

Subscription rate is $1.00 per year. Those interested make checks payable to "Clinical Proceedings Dept.," The Children's Hospital, Washington, D. C. Please notify on change of address.

Entered as second class matter November 21, 1946 at the post office at Washington, D. C., under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 538, Act of February 28, 1925, authorized January 17, 1947.

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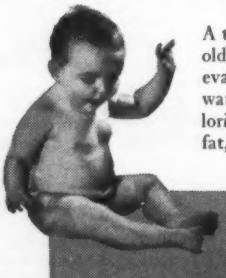

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THE SURGICAL TREATMENT OF FUNNEL CHEST

Marshall C. Sanford, M.D.

Funnel chest, also known as pectus excavatum, Thichterbrust, Chonechondrosternon, or costo-sternal depression, is a relatively common congenital, often familial, deformity which is amenable to surgical correction. A large number of the patients in whom it exists are severely incapacitated. This condition has long been recognized and much has been written about its etiology and treatment, but the importance of early surgical correction is not generally appreciated. In 1938 Ochsner and DeBakey[1] reviewed the literature on the subject and reported an operated case with a successful outcome. The following year Brown[2] described an operation for the condition, with several cases illustrating his work. Since then modifications in operative management have been proposed all working along the lines suggested by Brown in his original operative procedure.

Pectus excavatum has been described as a funnel-shaped depression of the lower end of the sternum and the adjacent costal cartilages. The apex of this depression is located at approximately the level of the junction of the xiphoid and the body of the sternum. Characteristically, this deformity becomes progressively more marked as the patient grows older. A paradoxical inward motion of the sternum is conspicuous on inspiration.

The etiology of the deformity appears to be due to a short central tendon of the diaphragm which puts this muscle under constant stretch, and with its contraction during inspiration, the lower sternum is pulled backwards. In time this produces a funnel-shaped costo-sternal depression. The lower costal cartilages are pulled inward along with the xiphoid and lower sternum to which the central diaphragmatic tendon is attached. There is no relation between this chest wall deformity and rickets since it is caused by an abnormal muscular pull, and not by the bony softening which results from a dietary deficiency.

The deformity is present at birth or is noticed in infancy and usually is progressive. There is little correlation between the extent of the funnel chest and the degree of disability thereby produced. The antero-posterior diameter or sternovertebral space becomes decreased and the chest appears thin and flattened. The dorsal spine becomes kyphotic and the patient takes on a malnourished appearance. Physical development is defective and most of the patients are underweight by the time adolescence is reached. They are extremely susceptible to respiratory infections and bronchiectasis is common. This is explained on the basis of defective bronchial drainage due to distortion and the retention of secretions resulting from ineffectual coughing.[7,8]

Lester has called attention to the allied thoracic deformities which are closely related to pectus excavatum since they, too, are produced by abnormal diaphragmatic pull. These deformities may be symmetrical or asymmetrical depending upon the portion of the central tendon which is

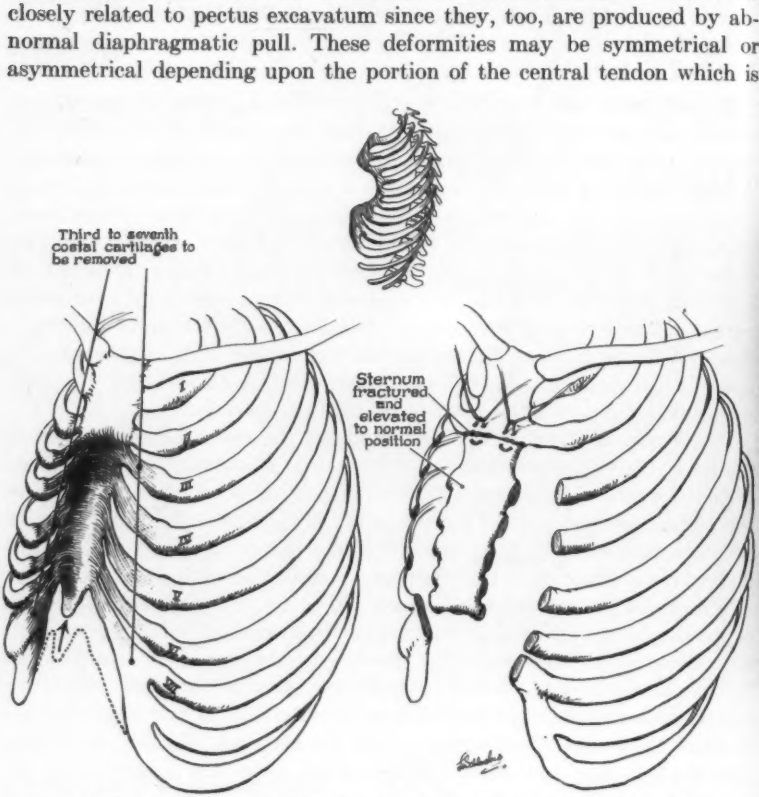

FIG. 1. The nature of the defect in a case of funnel chest with moderately severe deformity. The figure on the right shows the corrected position of the sternum. (*From Ravitch, M. M., Surgical Clinics of North America, October 1949, W. B. Saunders Co.*).

shortest. Asymmetrical deformities, obviously, are produced when one of the lateral leaflets is short. Transverse grooves similar to those encountered in rickets may be caused by a direct diaphragmatic pull on the costal arch. In the post-adolescent female, a funnel chest with its deep groove between the breasts may exaggerate their size, whereas an asymmetrical deformity in which one side of the chest is pulled inward causes the breast on that side to appear smaller. (Figure 2). Trauma and rickets may produce various thoracic deformities but they are unrelated to true funnel chest.

Ravitch[5] lists cosmetic, orthopedic, and physiologic indications for operation, while Lester[7] considers them under psychological and physiological headings. Actually the various indications are so intimately associated that differentiation is difficult. In many cases there are no symptoms in the early stages, but they appear as the child grows older. The infant who presents only an unsightly chest wall deformity when first seen may, if allowed to progress, return with irreversible bony changes and signs of cardiac and respiratory malfunction.

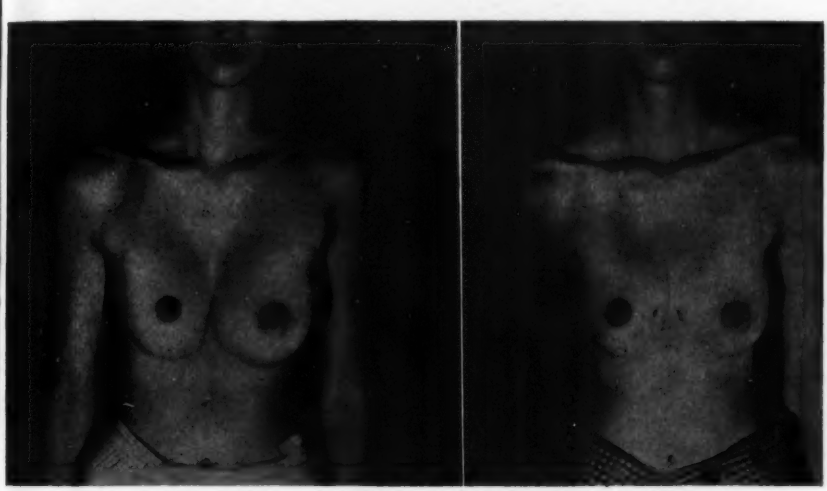

FIG. 2. A symmetrical deformity of funnel chest producing apparent underdevelopment of right breast corrected by operation. (*From Lester, C. W., J. Thoracic Surg., Vol. 19, April 1950*).

The deformity is unsightly and the sensitive child often resents the fact that he is different from others. He may refuse to participate in activities in which clothes do not hide his deformity. Psychological aspects increase in importance as the child becomes older. The deformity in girls attracts less attention until adolescence, at which time breast development magnifies it.

The changes that occur in the bony thorax are the factors responsible for the distortion and compression of organs within. There are all degrees of depression varying from a small indentation in the lower sternal region to a retraction which pulls the sternum back to, or *actually behind*, the ventral surface of the vertebral column. Bony changes increase with age and it is much easier to prevent them than to correct them once they occur. The deformity is reversible in the initial stages and early operation returns the

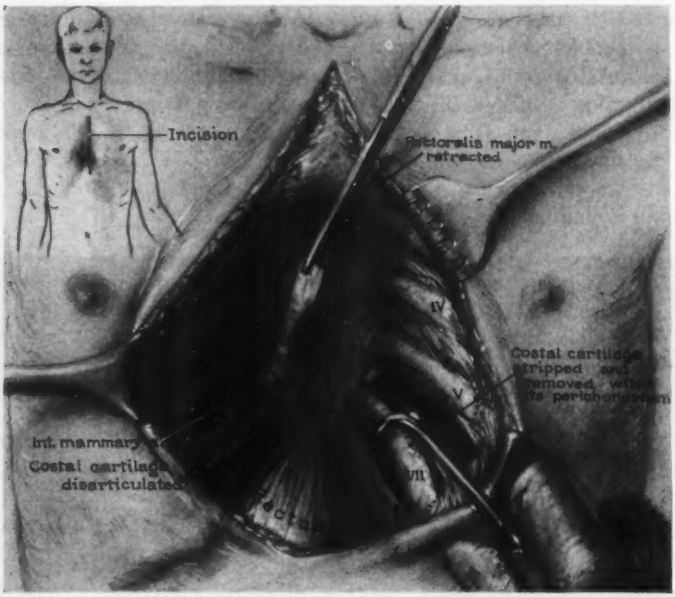

FIG. 3. Midline incision from manubrium to epigastrium. The pectoral muscles are stripped back and the costal cartilages resected together with the perichondrium. The entire deformed segment of each involved cartilage is resected. (*From Ravitch, M. M., Ann. Surg., Vol. 129, April 1949*).

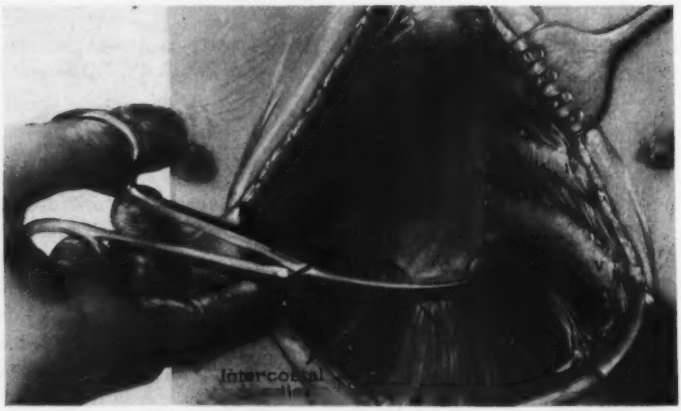

FIG. 4. The two lowermost costal cartilages on each side have been resected, giving access to the xiphoid. The xiphi-sternal articulation is divided. (*From Ravitch, M. M., Ann. Surg., Vol. 129, April 1949*).

thorax to normal. The physiologic disturbances resulting from this deformity are obviously the most important indications for surgical correction. Exercise tolerance is decreased and dyspnea is common. The true extent to which activity has been limited is seldom realized until after operation, when, for the first time, the patient experiences normal physiologic activity. Lung

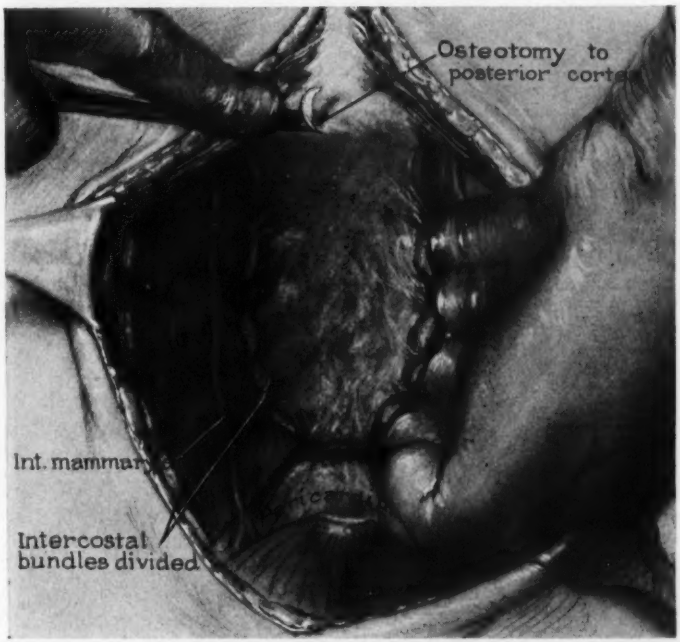

FIG. 5. Five costal cartilages on each side have been resected and the corresponding intercostal bundles have been divided. The sternum is freed except at its upper end. With the gouge a transverse osteotomy is performed. (*From Ravitch, M. M., Ann. Surg., Vol. 129, April 1949*).

volume is reduced by compression, and with the inward pull of the anterior chest wall, breathing becomes paradoxical, further reducing respiratory efficiency. The dyspnea is due primarily to cardiac restriction rather than to pulmonary compression. The heart is displaced to the left, and compression is produced which interferes markedly with cardiac dilatation. Angiocardiographic studies carried out by Dorner and his co-workers[9] suggest that the point of compression is in the region of the tricuspid valve. A systolic murmur is present in most cases and arrhythmia, precordial pain, and tachycardia are not uncommon. Some of the cardiac disturbances may be

explained on the basis of distortion and angulation of the great vessels. Much work is now in progress to determine the exact nature of the cardiac dysfunction.

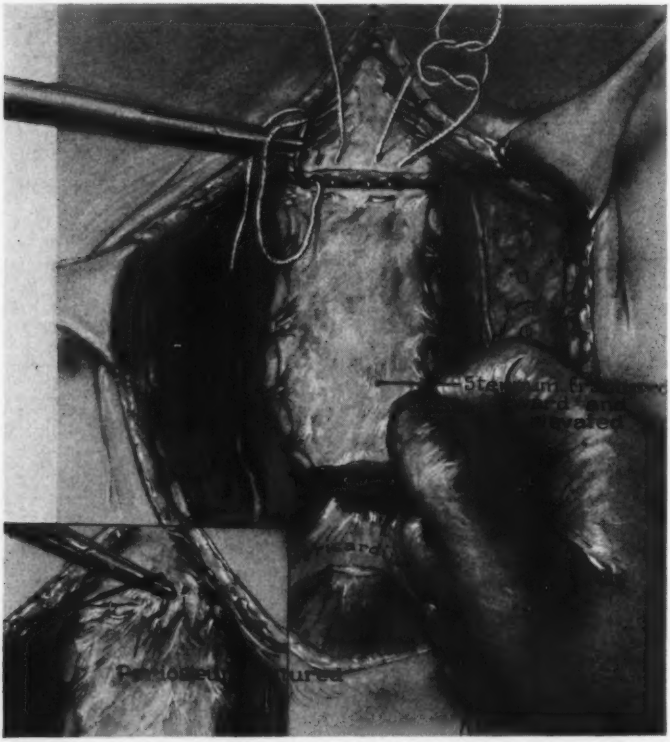

FIG. 6. The sternum is elevated anteriorly, fracturing the posterior plate at the level of the osteotomy. The corrected position is maintained by mattress sutures of braided silk placed through the bone. The periosteum is then sutured with black silk. (*From Ravitch, M. M., Ann. Surg., Vol. 129, April 1949*).

It is difficult for the physician to recommend operative repair of what appears to be an innocuous chest-wall deformity in a robust, asymptomatic infant or child who has never manifested any respiratory, cardiac or physiological disturbances. But to wait for these to appear may mean that a reversible condition no longer exists.

If the distortion is noted in infancy and increases or does not recede with age, operation should be performed to prevent irreversible deformity.

The earlier the operation is performed the better the result. The optimal time is between the ages of one and five years, for the deformity is reversible and

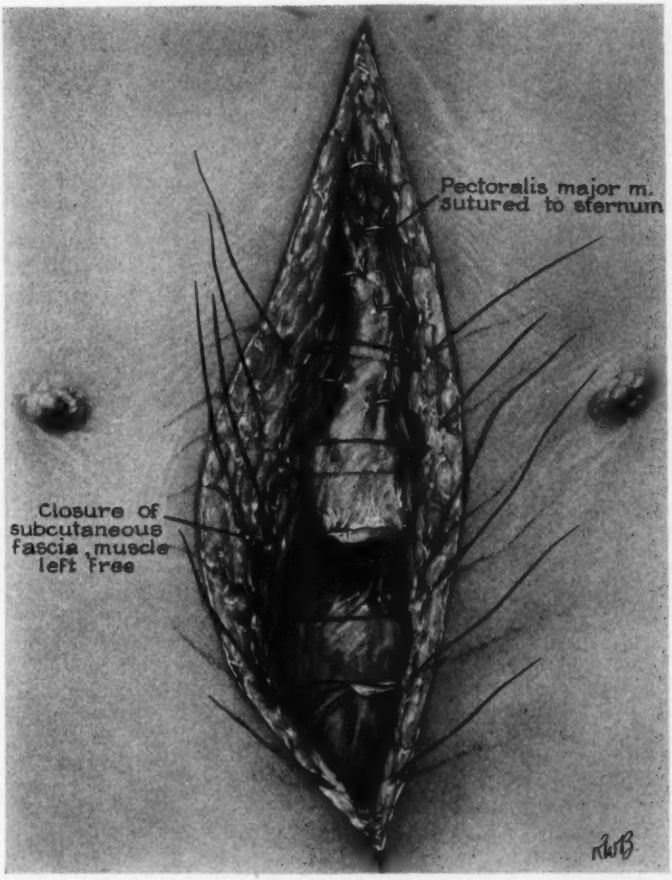

FIG. 7. The pectoral muscles are sutured to the sternum in the upper portion. No attempt is made to replace the xiphoid or to re-attach the recti. (*From Ravitch, M. M., Ann. Surg., Vol. 129, April 1949*).

excellent cosmetic results may be obtained since bony fixation of the deformity has not occurred. After the age of six, the functional results are good, although the cosmetic results may be disappointing. Lester believes that operation can be done at any age with the assurance that it will relieve

pressure symptoms, and probably will bring about at least an 80 per cent correction of the deformity.

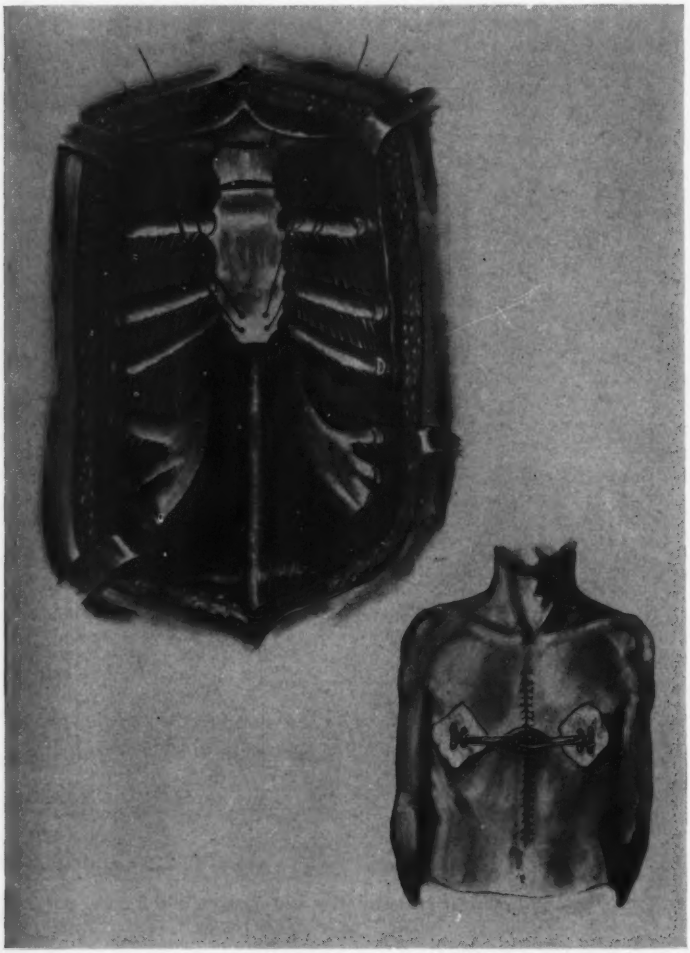

FIG. 8. Another method of surgical treatment illustrating a type of sternal immobilization. (*From Lester, C. W., J. Thoracic Surg., Vol. 19, April 1950*).

The operative technique may vary somewhat depending on the individual case, but the original procedure as outlined by Brown[2] and modified by Ravitch[5] and others[3,4,7] has proven quite satisfactory:

The deformed portions of all of the costal cartilage on both sides are resected, the xyphi-sternal articulation and substernal ligament are

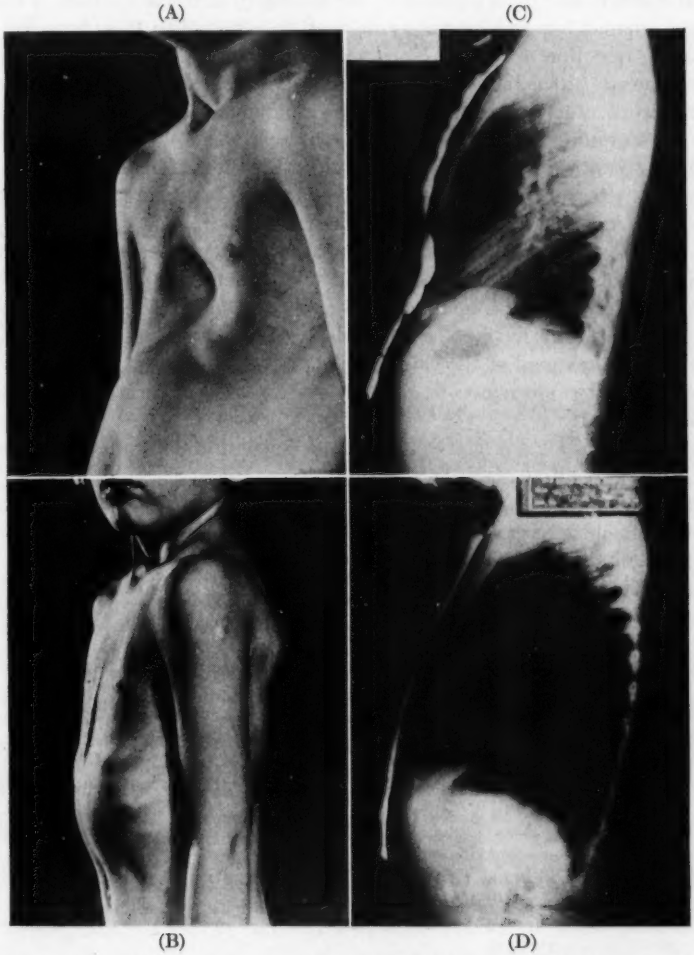

FIG. 9. A. Condition before operation. B. Condition three weeks after operation. C. Preoperative lateral roentgenogram. D. Postoperative lateral roentgenogram. (*Reported in Ravitch, M. M., Ann. Surg., Vol. 129, April 1949*).

divided, and the body of the sternum isolated except for its attachment to the manubrium. A transverse osteotomy at the superior border of the defect permits the sternum to be elevated to the corrected position which

is maintained by braided silk sutures in the bone. No external traction is employed and no cast or other support is required.[5]

If it seems wise in an occasional case to employ external fixation, a transverse angular bar or a cast may be used to accomplish this. In a few instances, resection of the deformed segment of anterior chest cage followed by reversal (turned upside down) and re-attachment has worked well. Some workers have recommended the employment of rib graft splints to fix the sternum in the corrected position.

Post-operative care includes the administration of *oxygen* by tent or nasal catheter for the first twenty-four hours. *Antibiotics* are given routinely since a severe infection in this area would be disastrous. A *portable x-ray of the chest* should be taken in the early post-operative period to rule out a pneumothorax, which may occur and escape detection during operation. *Skin sutures* should be removed late and one should be certain that any serum which may have collected under the skin flaps has been aspirated prior to the removal of the skin sutures.

SUMMARY

1. Funnel chest is a relatively common congenital deformity which may produce marked incapacity.

2. It is amenable to surgical correction, and the best results are obtained when surgery is performed early.

3. The indications for surgery are outlined.

4. Operative techniques and post-operative care are discussed.

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BRAIN ABSCESS IN A ONE MONTH INFANT

Case Report No. 206

Robert H. Parrott, M.D.
Sanford L. Leikin, M.D.
Hugh L. Ried, M.D.

We have recently encountered a case of brain abscess fatal to an infant at the age of one month, an occurrence rare enough in the medical literature to warrant special report.

CASE REPORT

F. W., a colored male, was admitted to the Children's Hospital on the service of Dr. William Burdick on October 2, 1950 and died sixteen hours after admission. This baby had been born of a full-term pregnancy and a labor that was said to have lasted three days, during which time the mother required sedation and intravenous fluids. There was no trauma at birth. The weight of the child was 6 pounds 2 ounces. He did well until his seventh day of life when he developed gasping respirations, hiccough, anorexia and a bout of apnea and cyanosis. He was admitted to Children's Hospital at this time and was found to have rapid shallow respirations. One spell, lasting 60 seconds, of apnea and cyanosis was observed during the initial examination. There was transient lateral nystagmus but no other eye signs, no meningeal signs or other evidence of neurological involvement. Examination of the heart and lungs was normal. Complete blood count and an x-ray of the chest were normal.

The child was placed in an oxygen tent and was given penicillin. The temperature remained normal throughout the hospital stay except for one rise to 100.4 F. There were no further episodes of respiratory distress or cyanosis during the thirteen following days when the child was observed in the hospital. He was apparently normal upon discharge.

The baby remained asymptomatic until a few days before the final admission, when he developed mild rhinorrhea and cough. At 4:30 p. m. the day of admission, he again showed cyanosis during a feeding and was rushed to the hospital. Physical examination then showed an acutely ill, minimally cyanotic, stuporous infant whose cranial sutures were widely separated and fontanelles bulging. His eyes showed no coordination. There was marked nuchal rigidity and a positive Kernig's sign. There was minimal rhinitis. The heart rate was grossly irregular, a systolic murmur was heard in the mitral area and a few fine rales were audible over scattered lung areas. The liver was palpable 6 cubic centimeters below the right costal margin. During the examination, the baby had a tonic convulsion with opisthotonus.

When a spinal tap revealed a slightly bloody fluid, which seemed otherwise clear, a ventricular tap on the right side was attempted. This yielded viscid, yellow-green pus in large amounts. Fifty cubic centimeters were immediately aspirated and penicillin was instilled. Despite sedation, parenteral fluids and parenteral administration of penicillin, sulfadiazine and aureomycin, there were repeated episodes of apnea, followed by opisthotonus and Cheyne-Stokes respirations. These symptoms were temporarily alleviated after a second aspiration of fifty cubic centimeters of pus but the baby remained comatose and expired sixteen hours after admission. The only laboratory examination performed was that of the aspirated pus which eventu-

ally showed *Escherichia intermedius* on culture. An autopsy was performed and the findings follow:

The body was that of a fairly well developed and well nourished colored male infant weighing 3.1 kilograms and measuring 50 centimeters in length. The fontanel was slightly sunken.

On gross examination the lungs did not completely fill the pleural spaces. They were greyish-red, and felt airless and firm. Cut section confirmed the homogeneous greyish-red appearance.

The liver and kidneys were grossly congested and the spleen and adrenals appeared normal.

On opening the cranial vault the venous sinuses were noted to be markedly dilated and engorged with blood. There was an area of recent hemorrhage in the posterior parietal lobe measuring 2 x 2 centimeters. In the left temporal lobe there was another recent hemorrhage extending from the lateral surface to the inferior edge of the lobe. This extended into the basal pathways with some attempt at resolution. The gyri were flattened, most markedly over the parietal area.

After formalin fixation, the sectioned brain revealed the presence of an abscess in the right occipital lobe. The abscess measured 5.5 x 2.5 x 5.3 centimeters. It had apparently ruptured through the body of the right horn into the ventricular system. The aqueduct was completely obstructed by a yellowish, gelatinous exudate. There was a thin capsule lining the entire ventricular system. The fourth ventricle was dilated and contained the same gelatinous exudate. An early abscess appeared to be developing in the left cerebellar lobe. Between the pons, medulla, and cerebellar lobes there was a plastic exudate measuring 3 to 4 millimeters in depth.

Histologically the lungs showed evidence of atelectasis. Many of the alveoli were collapsed while others were filled with erythrocytes, macrophages, and pink-fibrinous exudate. A moderate number of alveoli were lined with a pink hyaline membrane.

A moderate number of the glomeruli of the kidney showed atrophy of the tuft with dilatation of the capillaries. There appeared to be a moderate amount of pink staining debris in Bowman's capsule with compression of the glomerular tuft. Moderate dilatation of many of the convoluted tubules was present along with some purple staining material in the lumen.

Microscopic examination of the brain in the area of the abscess cavity showed a marked profileration of new small blood vessels which were somewhat fragmented. There was an increase in glial tissue, and an infiltration of lymphocytes, plasma cells, and a deposition of dark bluish staining material which appeared to be calcium. In some areas the number of polymorphonuclears predominated over the other types. In the area of hemorrhage a moderate thickening of the arachnoid with fibroblasts was noted. The number of astrocytes appeared to be increased in the subcortical layers.

The pathological diagnoses were:

1. Brain abscess, right occipital lobe, subacute
2. Subarachnoid hemorrhage
3. Pulmonary atelectasis
4. Glomerular atrophy and tubular dilatation due to obstruction, etiology undetermined.

DISCUSSION

This case is unusual for two reasons: 1) We have not been able to find a younger recorded case in the recent American literature or in two

earlier[1, 2] reviews of the subject; 2) The bacteriological etiology of brain abscess in infants is usually staphylococcal[1] whereas the organism in this case was one of the *Escherichia* group. The youngest patient with brain abscess previously included among the pathological reports of Children's Hospital was one who died at the age of 6 months. There was one case in the files of focal abscesses secondary to intracranial hemorrhage and necrosis in a 2 week old infant.

Baumoel[2] and his associates in 1942 were able to collect from all sources only 26 reported cases of brain abscess in infants. They do not mention any instance as young as the subject of this paper. Gauthier[3] reported a case from France of naso-ethmoidal progression of infection terminating in a fatal cerebral abscess in an infant one month old.

It is interesting but certainly inconclusive to speculate on the pathogenesis of this lesion in so young an infant. It would seem logical to assume that the episode of cyanosis at the age of seven days was due to the cerebral pathology and thus the onset of the abscess would have to be intragestational or intra-partem. The only clue available is the history of a prolonged labor, which might be construed as having been traumatic to the infant, even though the immediate delivery was not felt to be so. There was never evidence until the final admission of paranasal infection or otitis, felt to be the most common sources of brain abscess in children. There was no sign of urinary tract infection, gastrointestinal anomaly or infection of the umbilical stump to explain a source of the colon group organism. There was no congenital heart disease with arterio-venous shunt to allow the dissemination of infected thrombi to the brain. Direct implantation would have been impossible, unless some very pertinent detail of history was lacking. Thus we are left with the last of the four means by which Grant[4] feels a brain abscess can occur:

1. By direct implantation, as by a penetrating wound or surgery;
2. By direct extension from an adjacent source of infection;
3. By metastatic extension from a non-adjacent source through the blood stream;
4. By undetermined pathways from undetermined sources.

The diagnosis of brain abscess is infrequently made in infants before the terminal stages. This is readily understandable both from the rarity of the lesion and the paucity of early symptoms. The mortality is almost 100 per cent. If an early diagnosis were made, surgical drainage and antibiotics might lower this mortality. It would seem pertinent then to investigate fully for cerebral pathology when an infant presents with any unexplained symptoms such as the transient cyanosis and nystagmus in this case, which might be referable to early brain damage.

SUMMARY

1. A rare case of brain abscess due to *Escherichia intermedius* in a one month old infant is presented.

2. Possibilities of pathogenesis are discussed.

3. Clinicians are encouraged to investigate fully unexplained symptoms which might enable a diagnosis of brain abscess in infants before the terminal stages.

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MECKEL'S DIVERTICULUM COMPLICATED BY ULCERATION AND HEMORRHAGE

Robert O. Warthen, M.D.
Garnett W. Ault, M.D.

INTRODUCTION

Twenty-one cases of complicated Meckel's diverticulum have been seen in the Children's Hospital during the past twenty years (1930–1950). Five or 23.8 per cent of these twenty-one fell in the category of acute ulceration and hemorrhage.

Four of these cases will be presented in outline and one in detail. In addition, the clinical features of Meckel's diverticulum complicated by ulceration and hemorrhage will be discussed.

CASE REPORTS

No. 1. P. W., an eighteen-month old female, was admitted to the hospital twelve hours after passing a large bloody stool containing a few clots. A similar episode occurred shortly after admission, and two transfusions were required in preparation for surgery. There were no significant abdominal findings, and a flat plate of the abdomen and proctoscopic examination disclosed no abnormalities. Operation was performed five days after the onset of hemorrhage, and a preoperative diagnosis of an ulcerated Meckel's diverticulum was entertained. The operative procedure consisted of a diverticulectomy. The gross pathology indicated that the ulceration was located at the base of the diverticulum, and the microscopic examination revealed gastric mucosa. Uneventful recovery followed.

No. 2. G. R., an eight and one-half month old male, was admitted to the hospital twenty-four hours after passing an acutely exsanguinating bloody stool. During the following fourteen days two similar episodes occurred, and four transfusions were necessary in preparation for surgery. No significant abdominal findings were elicited, and a double contrast barium enema, a gastrointestinal series and a proctoscopic examination revealed no abnormalities. The preoperative diagnosis was "polyp." Operation was performed twenty-one days after the onset of hemorrhage, and a resection of the diverticulum and adjacent ileum with an end-to-end anastomosis was accomplished. The gross pathology disclosed an ulcer in the distal third, and gastric mucosa was identified microscopically. The recovery was uneventful.

No. 3. J. H., a five and one-half month old male, was admitted to the hospital three hours after passing a large, bloody stool. No other bleeding episodes occurred, and only one transfusion was required in preparation for surgery. There were no positive abdominal findings, and a gastrointestinal series was negative. No proctoscopic examination was recorded.

The preoperative diagnosis was ulcerated Meckel's diverticulum. Operation was performed five days after the onset of bleeding, and a simple diverticulectomy was performed. The ulcer was located at the base of the diverticulum, and microscopically was situated in ileal mucosa. An uneventful recovery ensued.

No. 4. R. S., a five-month old female, was admitted to the hospital twenty-four hours after passing a large, fresh, bloody stool with clots. Two similar acutely exsanguinating stools occurred during the next two weeks, and three transfusions were required in preparation for surgery. No significant abdominal findings were elicited, and the proctoscopic examination was negative. A gastrointestinal series and a barium enema revealed normal findings. Operation was performed twenty-two days after the onset of hemorrhage. The preoperative diagnosis was ulcerated Meckel's diverticulum. The operative procedure consisted of resection of the diverticulum and adjacent ileum and an end-to-end anastomosis. The ulcer was located near the base of the diverticulum, and the microscopic pathology revealed gastric mucosa. Uneventful recovery followed.

No. 5. V. F., a six-weeks old male, was admitted to the hospital one hour after completely soaking three diapers with fresh blood containing jelly-like clots. There had been no apparent colic, restlessness, or anorexia. The past history was noncontributory. The essential findings were an exsanguinated appearance and a rapid pulse. There was no evidence of petechiae or other hemorrhagic phenomena. The abdomen was soft, easily palpated, and no abnormality was noted. Proctoscopic examination to four inches, performed eight hours after admission, demonstrated dark, fresh, wine-colored blood on the mucosa and in the stool. There was no evidence of fissure, polyp, petechia, intussusception, or ulcerative colitis. The bleeding time was two minutes, the clotting time five minutes, and the blood platelet count 292,000.

The subsequent sequence of events is presented in chart 1.

The diagnosis of a polyp with slough and hemorrhage or a Meckel's diverticulum with hemorrhage from an area of ulceration was considered at the time of admission. The examinations and the persistent hemorrhage indicated that ulceration of a Meckel's diverticulum with impending perforation was the most probable diagnosis. Thirty-five hours after admission the abdomen was explored (G. W. A.) through a right midrectus incision, and the small intestine was examined from the ileocecal valve to the ligament of Treitz. A Meckel's diverticulum was located twelve inches from the ileocecal valve. The diverticulum, 2.5 centimeters long, arose from the lateral superior border of the bowel, and was adherent to the superior surface of the mesentery. A serofibrinous exudate covered its inflamed distal third and partially

CHART 1

Sequence of Events in Case No. 5

	HGB. GM /100 C.C.	RBC COUNT MILLION/CMM	WBC COUNT THOUSAND/CMM
On admission	11.0	3,470,000	
2 hours after admission	8.5	2,200,000	15,200
2½ hours after admission	Blood transfusion—90 cc.		
7 hours after admission	Hemorrhage—diapers soaked		
8 hours after admission	9.8	3,200,000	
9 hours after admission	Blood transfusion—100 cc.		
31 hours after admission	Hemorrhage—diapers soaked		
31½ hours after admission	10.0	2,300,000	5,000
32 hours after admission	Blood transfusion—100 cc.		
35 hours after admission	Blood transfusion—100 cc.		
	Operation		
6 days after admission	14.1	4,400,000	5,000

CHART 2

Comparison of Five Cases of Meckel's Diverticulum Complicated by Ulceration and Hemorrhage

	CASE 1	CASE 2	CASE 3	CASE 4	CASE 5
Mucosa of Meckel's diverticulum	Gastric	Gastric	Ileal	Gastric	Ileal
Location of ulcer in diverticulum	Base	Distal third	Base	Base	Distal third
Operative procedure: D = Diverticulectomy R = Resection & end-to-end anastomosis	D	R	D	R	R
Pre-operative diagnosis: M = Meckel's (ulcerated) P = Polyp	M	P	M	M	M or P
Time interval from onset of hemorrhage to operation	5 days	21 days	5 days	22 days	35 hours
Transfusions pre-operatively	2	4	1	3	4
Proctoscopic examination	Neg.	Neg.		Neg.	Neg.
Gastro-intestinal series		Neg.	Neg.	Neg.	
Barium enema		Neg.		Neg.	
Abdominal examination	Neg.	Neg.	Neg.	Neg.	Neg.

covered a thinned-out white spot representing an ulceration 0.3 centimeters in diameter. Perforation appeared to be the next pathological sequence of events. A thin mesentery containing an artery and vein extended for two-thirds of the length of the

diverticulum. After freeing the diverticulum from the bowel mesentery, 5 centimeters of bowel was resected in order to include the broad (1 centimeter in diameter) open base of the diverticulum. An open end-to-end anastomosis was performed. An uneventful postoperative course followed, and the child was discharged the eleventh day after operation. Normal bowel function has continued to date.

The gross specimen appeared to have ileal mucosa throughout except in the immediate vicinity of the ulcer. Here the mucosa was slightly nodular and irregular for 0.5 centimeters, and in the center a penetrating ulcer 0.3 centimeters in diameter could be demonstrated. The ulcer was directly over the distal portion of a branching vessel from the diverticulum mesentery. A small clot was present in the ulcer. It was felt that heterotopic tissue was present. However, diligent microscopic search for pancreatic, gastric, duodenal, colonic or other heterotopic tissue was unrewarding. The microscopic diagnosis was Meckel's diverticulum with acute ulceration of the ileal mucosa.

DISCUSSION

A review of the case reports presented and a survey of the literature indicate that Meckel's diverticulum complicated by ulceration and hemorrhage has certain clinical features that should be emphasized.

The outstanding clinical finding in these five cases was rectal hemorrhage of sufficient magnitude to soak the infant's diapers on one or more occasions. The discharge varied from fresh or wine-colored blood to jelly-like clots of blood. It was never tarry or tenacious such as one sees when hemorrhage from a duodenal ulcer occurs in infancy. It was not like the spotting or streaking of fresh blood along the stool that is seen in anal fissure of infancy and childhood. Hemorrhage of such magnitude can occur from the open end of a pedicle following torsion and slough of an adenomatous polyp of the colon. Proctoscopy with visualization of accessible polyps will usually solve this diagnostic problem; however, one may find it necessary to employ an air-contrast enema to locate them. On the other hand, if the lesion is not accessible, one can readily appreciate the difficulty of locating such a lesion at laparotomy. Hemorrhage of lesser magnitude may accompany intussusception, but the other clinical features of intussusception facilitate the differential diagnosis.

The second outstanding clinical feature of these five cases was the acute anemia requiring from one to four preoperative transfusions to bring the infant's blood picture to within normal limits. Most certainly anal fissure would not cause such blood loss. Duodenal ulcer and adenomatous polyp with slough and hemorrhage could produce such an acute anemia. Consideration of these conditions seems imperative in spite of the character of the bloody stool. In this respect it has been our experience that the amount of blood loss demands greater consideration than whether it is fresh, clotted, or partially digested.

The time interval between the onset of hemorrhage and the operation

is of significance in this small series. It is of major importance if one wishes to avoid an all too common perforation and/or severe chronic secondary anemia. The age of onset of the initial hemorrhage in the youngest infant was six weeks and in the oldest infant it was eighteen months. The time interval between the onset of hemorrhage and the operation was 35 hours in the youngest infant and 5 to 22 days in the other infants. The delay between the initial hemorrhage and the operation was properly utilized for supportive treatment and differential diagnosis. Gastrointestinal series was normal in three cases. It was omitted in two cases. Proctoscopy was performed in four cases with no demonstrable evidence of a polyp. Barium enema was performed in two cases with negative findings. These studies were of value in a negative fashion. We wish to emphasize that none of these patients were permitted to go for several months with repeated hemorrhages leading to a severe secondary anemia and/or the risk of perforation.

The presence or absence of heterotopia is of more than academic interest. Gastric mucosal patches were present in three cases, and ileal mucosa was present in two cases. The ulceration was located in the distal third of the diverticulum in two cases and at the base in three cases. In this small series heterotopia was present in 60 per cent of the cases that hemorrhaged.

We wish to comment upon the surgery performed in these five patients and to report that all had an uncomplicated recovery. Resection of the involved bowel was chosen as the procedure of choice in three cases (60 per cent) for it was felt that this would be the most thorough method of removal. In this respect we wish to call attention to the fact that ulceration is frequently present at the neck of the diverticulum or in the adjacent small intestine.[1-7] For this reason several authors have recommended resection of the contiguous ileum.[2,7,8] We concur in this latter recommendation. Diverticulectomy by simple amputation and closure was performed in two cases (40 per cent). This was considered a satisfactory method, and no complications followed.

CONCLUSIONS AND RECOMMENDATIONS

We wish to present a few conclusions and recommendations that may be drawn from a study of five cases of Meckel's diverticulum complicated by ulceration and hemorrhage, ocurring in infancy.

1. The diagnosis of an ulcerated Meckel's diverticulum should receive first consideration when acute hemorrhage from the rectum occurs in infancy. The hemorrhage is usually of sufficient magnitude to soak the diapers and produce an acute anemia. A presumptive diagnosis of hemorrhage from an ulcerated Meckel's diverticulum was made preoperatively in four of our five cases. This concurs with reports from others.[2,7,9,10] Other lesions that should be considered are duodenal ulcer and an adenoma or polyp

with slough. The bleeding encountered in anal fissure and intussusception is rarely of this magnitude.

2. The time interval between the onset of hemorrhage and the surgical treatment is of paramount importance if one is to avoid the all too common complication of perforation. Blood and fluid replacement are urgent measures that must not be omitted in preparation for surgery. Delay in operation invites extension of the ulceration so that perforation with an attendent increase in mortality is inevitable. We, therefore, respectfully urge that these infants be operated upon as soon as possible. We also recommend that these infants be proctoscoped, but that roentgen studies with barium be omitted.

3. We would suggest, without prejudice regarding other methods of removal, that resection of the ileum with end to end anastomosis be considered the surgical treatment of choice. This recommendation is made in view of the frequent occurrence of ulceration at the base of the diverticulum. Recent improvements in bowel management with respect to chemotherapy, antibiotics and intubation encourage us to make this recommendation.

SUMMARY

1. Five cases of Meckel's diverticulum complicated by ulceration and hemorrhage are reported.

2. The clinical features, differential diagnosis, pathology and surgical treatment are discussed.

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CLINICO-PATHOLOGICAL CONFERENCE

Directed by: E. Clarence Rice, M. D.
Assisted by: Richard J. Waters, M. D.
Sanford Leikin, M. D.
By Invitation: Morris Michael, M. D.

Richard J. Waters, M.D.

This white male infant was admitted to the hospital at the age of seven weeks and died on the thirty-first hospital day.

History reveals that the infant had vomited intermittently since the age of two weeks but had taken an evaporated milk formula eagerly between episodes. Three days before admission, the vomiting became constant after feedings and was almost projectile in nature. On several occasions cyanosis without dyspnea was noted after vomiting. The patient was seen in the out-patient department and admitted to the ward.

Past history reveals that the mother's pregnancy, labor, and delivery had been normal. The patient's birth weight was 8 pounds and 12 ounces and the immediate neonatal period was uncomplicated. The family history was non-contributory and this was the patient's first illness.

Physical examination on admission disclosed a thin dusky-appearing white male who was crying vigorously and whose lips and nails were moderately cyanotic. The rectal temperature was 100 degrees, the pulse was too rapid to count, and the respirations 32 per minute. Admission weight was 8 pounds and 3 ounces. The anterior fontanel was open and slightly depressed. The eyes, ears, nose, and throat were normal. The lungs were resonant to percussion and clear to auscultation. There was no cardiac enlargement, but a grade two systolic murmur was heard over the second left interspace. The heart sounds were of good quality, and no thrills were palpable. The abdomen was scaphoid, and the turgor of the skin of the abdomen was poor. No abdominal organs were palpable. The neurologic examination was negative.

The infant was given parenteral fluids immediately on admission in order to allay vomiting and dehydration. Cyanosis of the nail beds continued to be noticed on crying. A chest x-ray was reported negative and an EKG showed a slight degree of right axis deviation. The systolic murmur was not heard again.

On the third hospital day, the patient developed diarrhea, and for the remainder of his hospital stay he had from six to twelve loose green stools a day. He continued to take small amounts of fluid by mouth, but intravenous feeding was necessary almost continuously to maintain adequate hydration. Occasional bone marrow infusions were given when peripheral veins became thrombosed. Penicillin and streptomycin were employed in large dosage, and whole blood was given when necessary. Laboratory findings included urinalyses which were normal except for albuminuria in one specimen. All hemograms disclosed normal hemoglobin and erythrocyte levels and a constant leucocytosis of 15,000 to 20,000 white blood cells of which 60–80% were polymorphonuclear leucocytes and band forms while the remainder were normal lymphocytes. The temperature throughout the hospital stay was very irregular and varied between 95 degrees and 101 degrees almost daily. Weight loss was constant.

In spite of persistent therapy, the infant became moribund on the twentieth hospital day. Adrenal cortical extract was used to no avail and the patient expired 10 days later.

DISCUSSION

Morris Michael, M. D.: To paraphrase Sir William Osler, to understand diarrhea is to know half of pediatrics.

We have here a 7 week infant whose story was ushered in with vomiting and cyanosis and who was ushered out with severe diarrhea.

There is little other positive information except a constant leukocytosis, an apparently irreversible state of dehydration, acidosis, and continued course downhill.

Certain negative findings are of great importance: essentially normal heart findings, there being only a transient systolic murmur, negative chest x-ray in the presence of intermittent cyanosis, tachycardia, inability to palpate tumors, masses or organs in the abdomen. This child's early history from age 2 to 7 weeks was primarily one of vomiting, gradually becoming constant and almost projectile. No diarrhea is mentioned until three days after admission. With emesis of this order we think of:

1. Congenital malformations, especially of the gastro-intestinal tract, webs, partial atresias, or surgical conditions such as volvulus, intussusception, the latter more common in slightly older age groups.

2. Pyloric stenosis where constipation is the rule, although a starvation diarrhea may ensue. However in these groups we should have eventually distention of abdomen, palpation of a mass or x-ray evidence of an intra-abdominal catastrophe.

3. Neurological disorders, subdural hematomas, brain abscess, meningitis etc. which give increased intracranial pressure.

4. Any sepsis, severe infection in any location will lead to severe vomiting.

5. I dismiss as unimportant in this case functional vomiting seen so often in poorly handled infants, over-feeding, allergies and hypertonic infants seen so often in private practice.

The cyanosis which is so evident throughout must have its basis in: 1. Cardiac abnormality. 2. Lung pathology. 3. Intracranial injury. 4. Ingestion of nitrates or other poisons.

In severe cardiac failure I should expect to find at least a swollen liver, or some degree of heart enlargement. Atelectasis, if present was not of sufficient degree to show on x-ray or be heard on physical examination.

Intracranial disease has not been ruled out either by depressed fontanelle or a negative neurological examination. With ingestion of nitrites, as from well water, I would expect improvement after removal from previous environment.

The course of the diarrhea is typical of many with which we have struggled in the past. It is not necessary at this time to go into the many etiologies: virus, bacterial, parasitic, toxic, poisons, allergic, or so-called

parenteral diarrheas, intestinal infections which are secondary to another infection which the child is handling poorly. More rarely, diarrhea and vomiting may result in an infant with adrenal insufficiency. These are notably resistant to therapy, possibly because adrenal extract is not usually used in sufficient quantities.

Cystic fibrosis of the pancreas and its association with diarrhea and chest pathology is well known. In this case we have no chest findings although these are not at all necessary at this early age. We have also no confirmatory laboratory findings.

Thus, cyanosis in our case has been poorly explained by heart or lung pathology leaving only intracranial pathology or possibly an overwhelming sepsis.

The vomiting is poorly explained by intra-abdominal conditions, in absence of distention etc; possibly explained by intracranial pathology or severe sepsis.

The etiological agent of the diarrhea was certainly not discovered during life, and because of its later occurrence was probably a secondary infection in an already debilitated child.

Certainly we all remember mastoids which were drained, and myringotomies performed in hopes of finding the etiology in similar cases, usually to no avail.

Here again sepsis or intracranial pathology explain this symptom.

Meningitis of infancy may run a totally unsuspected course with none of the signs and symptoms usually seen, while vomiting, diarrhea and cyanosis due to increased intracranial pressure (blocking due to exudate) may predominate. I offer this as my primary diagnosis, with sepsis arising anywhere else in the body leading to a parenteral diarrhea, as my second diagnosis.

Certainly in all diagnostic problems of infancy a spinal tap should be done early.

PATHOLOGIC DISCUSSION

Richard J. Waters, M. D.: We are grateful to Dr. Morris Michael for undertaking the discussion of this patient, in whom the history and physical findings were suggestive of no specific clinical entity. Diagnoses entertained by the clinicians who attended the patient on the ward were: (1) diarrhea of undetermined etiology and (2) extreme malnutrition.

At necropsy the body was that of an emaciated white infant. The subcutaneous fat and muscles were practically non-existent. On opening the peritoneal cavity the liver extended 3 centimeters below the right costal margin and was dark red and firm with rounded edges. It was of normal weight. The organ cut with normal resistance and throughout the cut

surface were seen numerous, small, yellow, circular areas which were surrounded by dark red tissue. These were interpreted grossly as being small abscesses in various stages of development. The lungs were heavier than normal and on cut section numerous areas of patchy consolidation were seen. The mucosa of the small intestine and colon was light pink and smooth. The kidneys, heart, spleen and other organs were essentially normal.

Microscopically, sections of the liver disclosed numerous, small abscesses comprised of central zones containing masses of polymorphonuclear leukocytes and necrotic cellular debris. There was a moderate infiltration of inflammatory cells about the portal triads. Sections of the lung revealed marked peribronchial infiltration by polymorphonuclear leukocytes. The alveolar walls in these areas were thickened and the alveoli contained erythrocytes and a few "heart failure" cells. Other than for the presence of occasional plasma cells within the mucosa of the small intestine, sections of the gastrointestinal tract revealed no significant histo-pathological change. Histologically the remaining tissues and organs were normal.

The pathological diagnoses in this case were:

1. Multiple liver abscesses
2. Diffuse bronchopneumonia.

DISCUSSION

E. Clarence Rice, M. D.: This case was selected for discussion because it illustrates in the consideration of diarrhea of infancy, the possibility of certain factors, unrelated to a primary infection of the bowel, which cause the frequent bowel movements. The diagnoses in many of our clinico-pathological conferences have been rather unusual or uncommon, but such a condition as has been reported today is seen not too infrequently and is one that every student here should remember in the future.

Dr. Michael has mentioned the possibility of an otitic infection being the cause of a parenteral diarrhea and this has been noted by us on a number of occasions, likewise respiratory infections may be responsible for such a diarrhea, with no significant changes noted in the bowel wall. We can remember the unusual case of a newborn baby with pyloric stenosis, who following pyloroplasty, developed a diarrhea and subsequently was found to have fibrosis of the pancreas with duct dilatation. The fact that there are many other causes for diarrhea than an infection of the intestinal tract makes this case of practical importance.